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PRELIMINARY DRAFT

Functional and structural changes of glutamatergic receptors , increase the frequency of observing voltage as possible cortical gamma activator

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ABSTRACT :
In this presentation it is shown the way in which the structural and functional changes in a neuron in a standard state is changed to a HYPER EXCITED neuron, this causes an increase in voltage which can be in the EEG instrument shown as a gamma frequency in the region medium temporal lobe.
This complements the plausible hypothesis of cortical activators caused by NMDA glutamatergic receptor .
(*) (hypothesis put forward by Robert Moss in his article Gamma-band Synchronization & Cortical Columns).

Index :

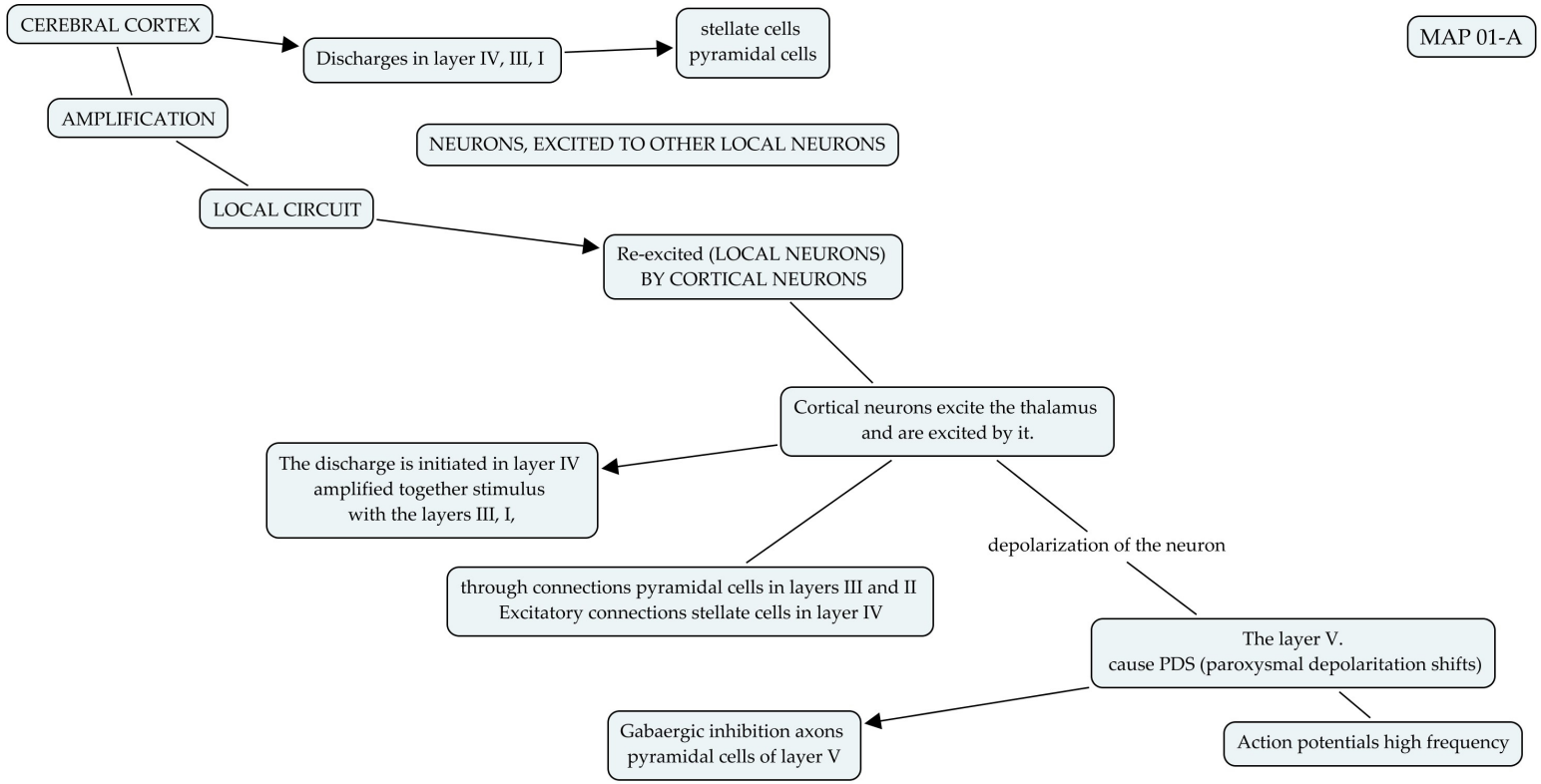
Hyperexcitable neuron (* 1):

The neuron state becomes normal hyperexcitable as a result of loss of inhibitory tone.

- Inhibitor Dismininución Gabaergic tone.
- Increased tone and activity of glutamate exciter amplifier circuits.

(* 1) Neuron hyperexcitable B + Increased exciter tone of glutamate, extracellular calcium C decreased, increased extracellular potassium glial fault therefore paroxysmal neuron discharge mode. This in stimulation of NMDA receptors N-methyl-D-aspartate receptor causing a sudden influx of sodium and slow calcium entry, the electric voltage is calcium dependent.
Repolarization by voltage-dependent channels potassium.

(*) Gamma frequency between 25 and 40 Hz is typical



Conceptual map :

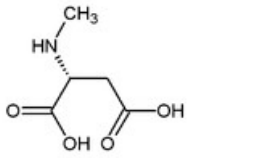
Re-excited local neurons by cortical neurons.
Cortical neurons excite the thalamus and are excited by it, the discharge is initiated in layer IV, amplified together stimulus with the layers (III and I), trough connections pyramidal cells in layers III and II, excitatory connections stellate cells in layer IV.

Deporalization of the neuron :

The layer V, cause PDS (paroxysmal depolaritation shits), action potentials high frecuency (gamma frecuency).
Gabaergic cells of layer V

Functional changes:

Increased concentrations of glutamic and decreased GABA concentration.
Increased sensitivity of glutamatergic receptor NMDA N-methyl-D-aspartate receptor and $GABA_A$ receptors decrease attributed to changes in phosphorylation and changes in the receptor subunits expressed consequent to changes in immediate genes.



(2*R*)-2-(methylamino)succinic acid

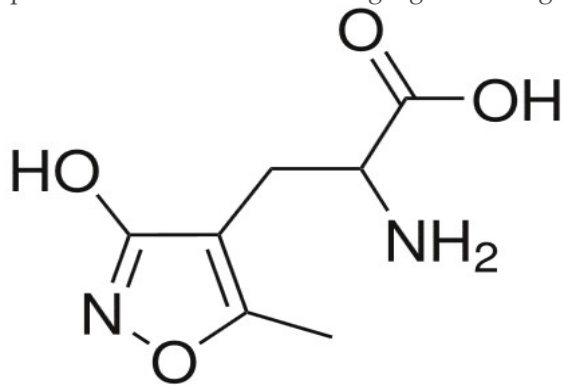
Structural changes:

Excessive activity of NMDA glutamatergic receptors , type 1 metabotropic N-methyl-D-aspartate receptor NMDA increase in intracellular calcium, loss of neurons by apoptosis, thus a consequence of activation of glutamic and calcium.
Shows that the increase in intracellular calcium caused by the sensitivity of NMDA glutamatergic receptors increases the voltage and the frequency is observed gamma
esctructural indicator of change. (possible cortical activator)

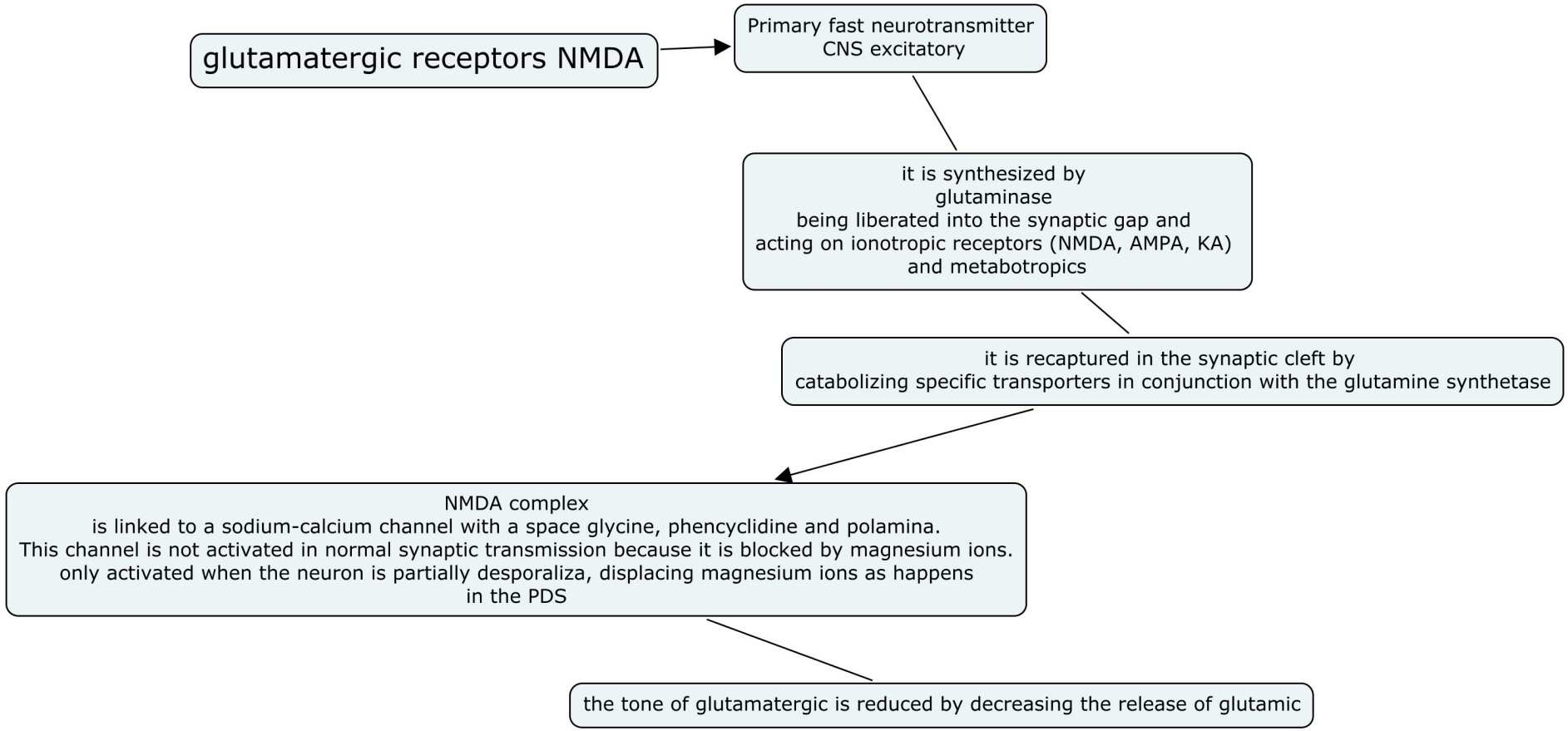
Changes paroxysmal depolarization:

(paroxysmal deporlarization shifts PDS) The PDS is initiated with a depolarization of the neuron that responds to high-frequency potential accompanied by a sustained depolarization that maintains the discharge, followed by a repolarization or hiperporalización neuron.

With the start of the discharge is attributed to activation-dependent sodium channel voltage glutamatergic receptors AMPA / KA



α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor AMPA / KA



Discharge paroxysmal T calcium channel low voltage (*hyperpolarize thalamocortical neurons)* **:**

Paroxysmal discharge is caused by the presence of calcium channel T low voltage which are activated when the neuron is hyperpolarized, it is caused by the action of GABA$_B$ *receptors, being stimulated by gabérnicas reticular neurons.*

When thalamocortical neurons are hyperpolarized effect of reticular neurons are activated by a hypersynchronic mode T calcium channel this causes a paroxysmal discharges onto the crust which is observed in an increase in the wavelength of the electroencephalogram, this is an activation of the bark with discharge corticotalámica reticular thalamic neurons, this results in an inhibition of the thalamocortical neurons, showing electroencephalogram wave.

Inhibition returns to hyperpolarize thalamocortical neurons, initiating a new wave-tip with 3-4 Hz frequency
An increased activity of a calcium channel or a possible GABA$_B$ *observed hyperfunction*

Conclusion of the hypothesis:

An ambivalent function is observed, in particular conditions is the variable chemical calcium into N-methyl-D-aspartate glutamatergic receptors NMDA which with increasing voltage can observe the gamma frequency, in other specific conditions are emotional variables which cause activation N-methyl-D-aspartate glutamatergic receptors.

We can infer hypothetical (until the end of the study), the cortical activator has the ambivalence of chemically cause neuronal hyperexcitability and simultaneously cortical emotions may be triggers, resulting in both situations the possibility of being seen with electroencephalographic analytical instruments.

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